In Fig. 4 there is a backward dislocation of the head of the radius, and as a result of this the head is much elongated and thinned. The impairment of motion occurs less sharply than in Fig. 5.

Fig. 5. The limitation here is due to the lack of development of the olecranon, which does not extend backward and upward to the normal amount. The result is that when the elbow is extended the tip of the olecranon impinges on the articular surface of the humerus instead of sliding up behind its lower end, and thus by actual bony contact prevents full extension. In this case the actual impairment of movement appears increased to the observer on account of the curving forward of the lower end of the shaft of the humerus, which in addition to the real impairment produces an apparent flexion.

Fig. 6 is another excellent radiogram of an elbow in this affection. There is some lateral dislocation and the bones hitch on extension.

Fig. 7 is a radiogram showing both the shoulder-joint and the elbow-joint. There is marked atrophy of the bones of the upper extremity, and in this case there was a posterior subluxation of the head of the humerus.

Fig. 8 shows anterior subluxation of the head of the humerus, which is about the normal size and is to be compared with

Fig. 9, which shows marked atrophy of the head of the humerus in a case with posterior subluxation, and retarded development of the epiphysis.

Fig. 10 is a radiogram of another case of posterior subluxation, showing the dropping of the head of the humerus from the scapula on account of paralysis of the deltoid and stretching of the capsule from the weight of the limb. This is from the same patient whose elbow is shown in Fig. 5.

TACTILE AMNESIA.

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Not infrequently patients suffering from hemiplegia are unable to recognize by touch objects in the paralyzed hand. This must occur if there is complete insensibility to all forms of sensory stimuli. But it also occurs when there is only partial insensibility, when only certain kinds of sensations are unperceived. Mere tactile anesthesia —that is, the inability to feel contact on the skin without pressure or at least with no more pressure than is made by a wisp of cotton— does not prevent the recognition of objects. When the anesthesia affects the underlying tissues so that deep pressure is not felt, the patient not only does not know what he has in his hand, but does not know that he is grasping anything. More important, then, than light touch in the recognition of objects is the ability to know the position of the fingers, to differentiate weight, to locate the point touched, and to recognize the number of points of contact. Failure in these regards, even if mere touch sensibility is preserved, leads to true astereognosis. In healthy men objects are distinguished much more accurately by handling them than by contact with any other part of the body. Some objects (apart from recognition by taste) can be recognized by contact with the lips, tongue, and mucous membrane of the cheeks. The ability to recognize objects in contact with the feet is almost *nil*. This is probably largely owing to the fact that throughout life no attention is given to educating the sensibility of the feet and partly to the lack of prehensile power in the toes. Armless men who have learned to use their feet more or less well as their hands have a greatly developed power of recognition of objects by them. Persons who have been hemiplegic from birth or early childhood are sometimes astereognostic in the palsied arm from lack of training.

These few preliminary words about astereognosis are necessary because it is often confused with tactile amnesia. In the latter sensibility to all forms of stimuli is preserved, but the patient, though knowing he has something in his hand, yet cannot recognize what it is. It is not that he cannot, though knowing what the object is, recall its name (tactile aphasia), but he does not know its nature or uses, knows nothing about it. Of course, the recognition of an object is a psychical process. A great number of sensations from the skin and underlying structures, muscles, and joints are combined and form the perception of a key, an apple, or whatever the object may be. Though the judgment is psychical there must be stored, somewhere in the cortex, memories of passed tactile sensations of other keys, apples, etc. Disease destroying this part of the cortex or cutting it off would lead to tactile amnesia. The location of this centre is yet not clearly and definitely known, but it probably is in either the posterior part of the parietal lobe or the anterior part of the convex surface of the occipital lobe. The frequency of the association of the symptom with word blindness and mind blindness makes it probable that the centre is near the higher visual centre. It cannot be identical with the centre for common sensation, because tactile sensibility is not affected. The frequent occurrence of the symptom with mixed sensory aphasia due to a unilateral lesion would indicate that the centre is unilateral. It must be posterior to the fissure of Rolando. The symptom never occurs if the area of disease involves only the prefrontal lobe. I have never seen it in pure motor aphasia, in uncomplicated left-sided hemiplegia, nor in right-sided hemiplegia unless there was also a greater or less degree of word blindness or object blindness or some type of sensory aphasia. It is frequent in pure mind blindness and in mind blindness occurring as an early symptom of dementia caused by diffuse organic brain disease. There may, however, be complete mind blindness, an absolute inability to recognize any object by sight, without any tactile amnesia.

In differentiating it from astereognosis it must be remembered that there is no disturbance of primary sensibility and that both sides are affected. I have never seen it as an entirely isolated symptom and do not know whether it occurs. It is sometimes an early or late symptom of organic dementia. Its presence or absence cannot of course be determined in severe cases of total or of mixed sensory aphasia, because it is impossible to make such patients understand what they are to do when an object is put in the hand. The only patients suitable for examination are those in whom the aphasia is partial or in whom recovery has gone far enough for the patients to understand short and simple commands and to use language enough to show whether they know what they are grasping. Nothing can be judged from the patient's general conduct whether the symptom is present or not, because vision helps him in all he does. It can only be discovered by examination.

CASE I.—*Complete tactile amnesia associated with sensory aphasia, hemiplegia, word blindness, and partial object blindness.*

J. M., seventy years old, was admitted to the hospital in December, 1902, with right hemiplegia and jargon speech. His previous history could not be obtained, but he must have been ill quite a long time, because the paralyzed arm was contractured and his gait was that of an old hemiplegic. Spontaneous speech, that is, speech as a method of expressing ideas arising within his mind and not merely words recalled reflexly in response to external stimuli, was an unintelligible jargon. There was not complete word deafness. He understood and obeyed short and simple verbal commands such as "stand up," "sit down," "walk," "stand still," but failed to obey any order in the least complicated. His difficulty seemed to be not so much in understanding single words as in carrying the first words of a long sentence in consciousness until the last were spoken. Words made only a momentary impression on him. He understood gestures and pantomime fairly well. By the combined use of spoken words, gesture, and pantomime I was able to make him understand he was to tell me the names of objects shown him and put in his hands. He named at sight a box, spoon, fifty-cent piece, ten-cent piece, watch, book, penholder, candle, and scissors. He could not name and gave no evidence of recognizing a fork, cigar, key, or knife. He failed to recognize any object put in either hand. He did not understand written or printed words and could not even write his name. His condition remained unchanged for two months, when he died of chronic Bright's disease.

CASE II.—*Transitory hemiplegia and permanent jargon aphasia with partial tactile amnesia.*

J. C., seventy-six years old, was admitted to the hospital in January, 1906. Two and one-half years before he had had a stroke, causing transitory difficulty in speech. The day before admission he fell unconscious and so remained a few minutes. When consciousness returned speech was so mixed that he could not be understood and he was palsied on the right side On admission speech was so mixed that it was impossible to understand him. He was not entirely word deaf because he obeyed simple verbal commands, but he failed to obey any complicated order. There was no hemiplegia. Three days later he had improved a great deal. He wrote his own name, but nothing else. (He had been taught to read and write.) He looked blankly at written and printed words and made no attempt to obey written orders. He named at sight most numbers below 100, but failed on all larger ones. He recognized and named all the letters except U. He copied a few short words, but soon became confused and ceased to make any further effort. He did not understand the meaning of the words he copied. Spontaneous speech had improved very much. He could carry on a simple conversation for a few minutes, but soon became mentally tired and confused and then his speech became very mixed. He recognized and named at sight almost all the common objects shown him. Some objects put in either hand he recognized and named; some he recognized, as was proved by his description of them, but could not name (tactile aphasia); others he did not recognize. Some objects recognized by touch were not recognized by sight, and vice versa. There was no anesthesia of any kind in either hand. When he was discharged several weeks later spontaneous speech was quite good, but he still became confused if he talked for a long time. His disability in recognizing objects by touch and his inability to read and write remained unchanged.

CASE III is very complicated because in the paralyzed hand there was sufficient sensory disturbance to produce astereognosis, while in the left, with normal sensibility, the patient was able to recognize and name some objects but not others—partial tactile amnesia.

C. D., forty-five years old, was admitted to the hospital in April, 1905. Two months before admission he suddenly became confused, mixed in speech, and, in the course of a few hours, lost power in the right arm and leg. He had a right-sided hemiplegia. He could walk, but dragged the right leg, and the right arm was contractured. There was slight palsy of the lower right face. He had marked disturbance of spontaneous speech. On beginning to talk he did well, but soon tired, became confused, and then speech was unintelligible. He understood and obeyed simple verbal commands and answered simple questions by simple words. He became much mixed on attempting to use long sentences in talking. He recognized and named most objects at sight; a few he described by a paraphrase or a not strictly accurate name, that is, a teaspoon was a "thing to eat with" and a cork was a "stopper." He recognized by sight all the letters and could read aloud and understand the meaning of short words. He could copy and sometimes understand words he copied. He recognized numbers of not more than two figures. He could write his signature C. B. Blank, but not his first name, Charles. He could write most letters at dictation, but no words. He could not recognize any object put in the right hand. Though the right arm was palsied, there was enough power in it to grasp and "actively" feel objects. Sensibility to touch was preserved, but though he located a single point of contact fairly well on the right hand he could not distinguish whether he was touched in one, two, or three places simultaneously unless they were very wide apart. When touched in several places at once he usually located one fairly well, but apparently had no knowledge of the others. He knew in a way, but not accurately, the position in which the fingers of the right hand were passively put, because he could put the fingers of the left hand in about the same attitude. On the left hand there was no disturbance of sensibility of any kind. In it he recognized a few objects, but failed entirely to recognize many.

CASE IV.—*Slight hemiplegia, mixed sensory aphasia, partial object blindness, and partial tactile amnesia.*

S. B., fifty years old, was admitted to the hospital in April, 1904. He had an old right-sided hemiplegia involving the face. He could walk a little and move the right arm slightly, but not use it for any purpose. On admission his speech was such a jargon that it could not be understood, and he obeyed no verbal commands. By the third day he had improved enough to understand and obey simple commands. He understood that he was to tell me what the things were I showed him and put in his hands. At sight he recognized and named a watch, handkerchief, one-cent and five-cent pieces, two and five-dollar bills (as money, but not the denomination), a spoon, a cigar, bunch of keys, and a cup. Many other common objects he did not recognize. By touch in the right hand he recognized and named a penknife, key, and watch, but nothing else. In the left hand he recognized nothing. If he handled and looked at an object he did not recognize and at the same time was told its name he did not recognize the name. Speech improved a great deal, but his visual and tactile disturbance continued until death, which occurred some weeks later from uremia.

CASE V.—*Transient right hemiplegia, aphasia, and tactile amnesia.*

W. C., sixty years old, was admitted to the hospital in August, 1904. On January 14, 1904, he awoke paralyzed on the right side and with inarticulate speech. He improved, but the day before admission speech again became affected. He had never been taught to read and write. The lower right face was slightly palsied. At the beginning of the examination he answered questions without difficulty, but soon tired mentally and became confused and misused words. There was no disturbance of sensation. He recognized and named almost all objects shown him, but could not recognize or name any object put in either hand. In a few weeks he could talk fairly well and recognized and named all objects by touch. He died some months later without any return of palsy or aphasia.

CASE VI.—*Marked dementia, mind blindness, slight amnesic aphasia, and partial tactile amnesia.*

J. M. was an educated man, fifty-seven years old, and had been actively engaged for years in literary pursuits. He had been a heavy drinker, but so far as known had never had syphilis. Early in 1898 he began to be irritable and easily angered and careless about his business affairs, but nothing was suspected of any serious illness until July, when he was suddenly taken ill, becoming delirious and vomiting frequently. He continued to be very excited for several days and then became quiet, but his speech was "a mere gibberish." He was not paralyzed and his speech slowly returned to normal. A similar attack recurred in September. He was alleged to have been blind after both attacks for a short time, but it probably was mind blindness, for he avoided obstacles in walking. He never worked after the first attack, lost all ambition, and was easily controlled.

His general nutrition was good. Gait and station were normal. There was no paralysis of the extremities or cranial nerves. Both knee-jerks were spastic. His voluntary speech was somewhat affected. For the most part he spoke well, but occasionally he would fail entirely to recall a word, or would misuse a word or be compelled to make a paraphrase. He recognized his errors immediately. He understood all that was said to him, but after conversing for some minutes would become a little confused and then his speech would become much worse. He could repeat any word said to him. He could write a few words or short sentences from dictation equally well with the eyes open or shut, but could not read what he had written. Writing soon fatigued him and then he failed entirely. He could not read any writing or print. He could not recognize any object by sight, but knew there was something before him and whether it was longer than broad, or whether it was larger or smaller than some other object shown him at the same time. He recognized the nature and uses of a few objects on handling them, and described them by paraphrase, but could not recall the names. The great majority of objects he did not recognize by touch at all. There was no disturbance of sensibility in either hand. Dementia at first slight, later became quite marked. He had then no realization of the fact that he was a seriously ill man. On the contrary he was overhappy, but had no definite delusions of grandeur. He had lost all intellectual interests. There was a double murmur at the aortic cartilage, hypertrophy of the heart, and the radial and temporal arteries were much thickened. He had no subjective symptoms of heart disease. The most accurate diagnosis of his case is organic dementia caused by diffuse cerebral degeneration. Though the mind blindness, aphasia, and tactile amnesia increased his dementia, they were not its primary cause, because it began before they appeared.

CASE VII.—*Word blindness and partial tactile amnesia coming on after a sudden hemiplegia and followed by complete dementia, caused by arterial sclerosis and brain tumor.*

The patient was a merchant, fifty-five years old, who about nine months before I first saw him suddenly became hemiplegic. He was a fairly well-built, elderly man. His gait was slightly paralytic and at the same time spastic. There was not marked hemiplegia, but slight athetoid movements were constant in the right arm and were made worse by any voluntary muscular effort. Both knee-jerks were increased, the right being the greater. The right grip was weak, the left good. Babinski jerk was present in the right foot. Speech was normal and he understood everything said to him. He could not understand writing or print except single letters and a few short words. He recognized and named all but a very few objects at sight. He could recall the names of only a very few, though he recognized them. He could recognize and name a few objects by touch; others he recognized, but could not name; but by far the greater number he neither recognized nor named. He could not write with the right hand and any attempt to do so greatly increased the athetoid movements. With the left hand he could write many letters and a few short words. In the left hand sensibility was normal, but in the right two points of contact had to be widely separated to be distinguished. The right pupil did not react to light, the left reacted feebly; both reacted well with convergence and accommodation. There was quite marked optic atrophy on both sides.

A few months later he had a general epileptiform convulsion, and several weeks later awoke one morning speechless except for the words "yes," "no," and "doctor." In the course of a few days speech returned. Several months later he had six general convulsions in one day. Once he had an attack of transitory blindness and jargon speech. As time passed he grew weaker on the right side and his mental state deteriorated. His memory became very poor. He failed to recognize relations and friends, sank into complete dementia, and died.

At necropsy a large tumor was found infiltrating the whole left parietal lobe and the anterior part of the occipital lobe. It did not pass the fissure of Rolando anteriorly. Microscopically it proved to be a sarcoma. The cerebral arteries were all very atheromatous, and many small areas of softening were present in both hemispheres. There was mitral and aortic valvular disease, hypertrophy of the heart, and chronic interstitial nephritis. The lesions were so widespread that the case could not be used to locate any function.